Cerebral Venous Thrombosis

Anna Darby, MD, MPH[a,b], Megan Bocchicchio, MD[c], Ramin Tabatabai, MD, MACM[a,c,*]

KEYWORDS

- Cerebral venous thrombosis • Venous stroke
- CT venography (CTV) & MR venography (MRV) • Hypercoagulable states
- Headaches • Seizures

KEY POINTS

- CVT is a rare but serious cerebrovascular condition that remains difficult to diagnose due to its variable presentation and limited detection on standard brain imaging.
- Risk factors for CVT include hypercoagulable states, hormonal influences, infections, neurosurgical procedures, and emerging associations such as SARS-CoV-2 and vaccine-induced thrombocytopenia.
- Clinical presentation varies widely, with headaches being the most common symptom, followed by seizures, focal neurologic deficits, and altered mental status.
- Diagnosis relies on neuroimaging, with computed tomography (CT) venography and Magnetic Resonance (MR) venography being the most sensitive modalities.
- Management includes anticoagulation therapy with direct oral anticoagulants emerging as a viable alternative to traditional heparin bridging; severe cases may require neurosurgical intervention.

INTRODUCTION

Cerebral venous thrombosis (CVT) is a rare yet significant cerebrovascular condition, distinct from ischemic arterial stroke in both vascular distribution and clinical presentation. Despite being a serious cause of secondary headaches in the emergency department, CVT remains difficult to identify due to its low prevalence, variable presentation, and suboptimal yield on standard radiographic brain imaging.

In the United States and globally, CVT remains a rare diagnosis, accounting for approximately 0.5% to 3% of all strokes.[1–5] However, recent registry data indicate a rising incidence of CVT cases since 2010, likely due to increased clinical awareness and advancements in diagnostic imaging.[6]

[a] Department of Emergency Medicine, USC Keck School of Medicine; [b] Los Angeles General Medical Center, Los Angeles, CA, USA; [c] Department of Emergency Medicine, Los Angeles General Medical Center, 1200 North State Street #1011, Los Angeles, CA 90033, USA
* Corresponding author. Department of Emergency Medicine, Los Angeles General Medical Center, Los Angeles, CA.
E-mail address: rtabatabai@dhs.lacounty.gov

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Abbreviations	
AHA	American Heart Association
CTV	CT venography
CVT	cerebral venous thrombosis
DOACs	direct oral anticoagulants
ICP	intracranial pressure
LMWH	low molecular weight heparin
LP	lumbar puncture
MRV	MR venography
NCCT	noncontrast computed tomography
VITT	vaccine-induced thrombotic thrombocytopenia
VKAs	vitamin K antagonists

This review highlights updated evidence, including the 2024 AHA scientific statement on CVT, to aid emergency clinicians in optimizing diagnosis and management of this complex condition.[1]

DISCUSSION

Pathogenesis

CVT occurs when a thrombus forms in the superficial cortical veins or dural venous sinuses, leading to increased pressure within the cerebral venous system.[7] (**Fig. 1**) Isolated cortical venous thrombosis is rare, with only 116 reported cases in the literature. An isolated case of cortical thrombosis typically presents with focal neurologic symptoms and is less commonly associated with elevated intracranial pressure (ICP).[8,9] However, most cases extend into the dural venous sinuses increasing the

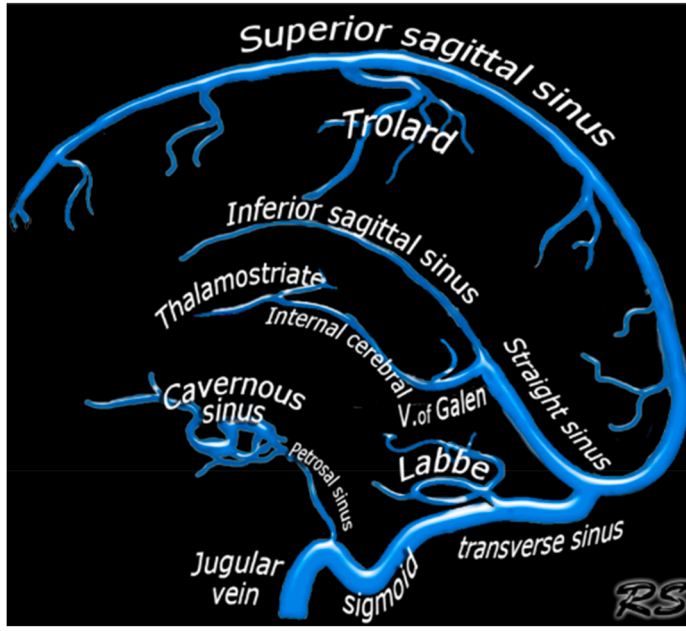

Fig. 1. Cerebral venous system (*C*) RS. (Cerebral Venous Sinus Thrombosis. Retrieved from: https://radiologyassistant.nl/neuroradiology/sinus-thrombosis/cerebral-venous-thrombosis.)

risk for elevated ICP. The resulting physiologic effects include blood-brain barrier disruption and vasogenic edema, which can progress to cytotoxic edema, ischemia, and elevated intracranial pressure due to impaired cerebrospinal fluid absorption. Presentation can vary based on the thrombus location (**Table 1**); however, the anatomic location does not reliably produce consistent, reproducible clinical syndromes.[8]

A crucial strategy in addressing CVT is ensuring emergency clinicians recognize its risk factors (**Box 1**). Over 85% of CVT patients have at least 1 identifiable risk factor, emphasizing the need for thorough history-taking.[10] Women, particularly those of reproductive age, have traditionally been considered at higher risk due to hormonal and reproductive factors.[11–13] This increased incidence is primarily linked to gender-specific contributors to hypercoagulability, such as oral contraceptive use, hormone replacement therapy, pregnancy, and the puerperium period. However, recent data suggest a shift in demographic patterns, with males now affected nearly as often as females, potentially due to a declining incidence of postpartum CVT.[6]

Other significant risk factors for hypercoagulability include inherited or acquired hematologic disorders. Additionally, localized head and neck infections, such as Lemierre's syndrome or mastoiditis, as well as recent neurosurgical procedures such

Table 1
Cerebral venous thrombosis presentations by anatomic location

Affected Dural Sinus	Clinical Presentation
Superior sagittal sinus (25%–45%)	• Headache (most common) • Seizures (often focal) • Motor deficits (variable) • Alteration of mental status • ICP elevation (papilledema, nausea, and vomiting)
Transverse sinus (25%–60%)	• Headache • Ear or mastoid pain • ICP elevation (papilledema, nausea, and vomiting)
Straight sinus (15%–18%)	• Headache • Alteration of mental status • Encephalopathy • Coma (severe cases) • ICP elevation (papilledema, nausea, and vomiting)
Sigmoid sinus (5%–15%)	• Headache • Ear or mastoid pain • Pulsatile tinnitus • Lower cranial nerve deficits (dysphagia and hoarseness)
Isolated cortical veins (rare)	• Headache • Focal neurologic deficits (aphasia, confusion, and hemiparesis)
Cavernous sinus (often septic source)	• Regional syndrome of periorbital and forehead pain • Proptosis • Ocular chemosis • Cranial nerve palsies III, IV, VI, and the ophthalmic (V1) and maxillary divisions (V2)
Deep venous system for example, internal cerebral veins, vein of Galen, basal veins of Rosenthal (10%)	• Alteration of mental status • Rapid neurologic deterioration • Drowsiness, stupor, or coma

Box 1
Cerebral venous thrombosis cerebral venous thrombosis risk factors

Risk factors
- Hormonal factors
  - Oral contraceptive use
  - Hormone replacement therapy
  - Pregnancy & postpartum period
- Hypercoagulable states
  - Factor V leiden mutation
  - Prothrombin gene mutation
  - Protein C or S deficiency
  - Antithrombin deficiency
  - Antiphospholipid syndrome
  - Malignancy
  - Myeloproliferative disorders
- Infections and inflammatory states
  - Head & neck infections (eg, sinusitis, mastoiditis)
  - Systemic Inflammatory Disorders (e.g. lupus, IBD)
  - Covid-19 infection
- Mechanical & procedural risks
  - Neurosurgical procedures
  - Jugular venous catheterization
  - Trauma or head injury
- Other Medications & Vaccines
  - *Adenoviral-vectored* Covid Vaccine (AZD1222 AstraZeneca & AD26.COV2.S Johnson and Johnson/Janssen)
  - Glucocorticoids, tamoxifen

as craniotomies or sinus procedures with proximity to dural venous sinuses, jugular venous catheterization, and space-occupying lesions compressing the dural sinus, can contribute to CVT.[1] More recently, obesity, COVID-19 infection, and vaccine-induced thrombocytopenia have emerged as notable risk factors.[1]

Clinical Presentation

CVT presents with diverse clinical manifestations and is frequently misdiagnosed initially, underscoring the need for early recognition to ensure timely diagnosis and management. The clinical presentation, though variable, largely depends on the anatomic dural sinus location of the venous clot and the extent of resulting ischemia and intracranial hypertension (see **Table 1**).

Key symptoms of CVT include headaches, seizures, focal neurologic deficits, and altered mental status. CVT should be considered in the differential diagnosis for patients presenting with these findings, particularly in patients with identifiable risk factors.

Headache is the most common symptom of CVT, occurring in up to 90% of patients.[14,15] The headache is typically persistent and progressive over hours to weeks and may be unilateral or bilateral.[16] Less commonly, patients experience a thunderclap headache, mimicking the presentation of subarachnoid hemorrhage and other thunderclap-related etiologies.[17] Symptoms that worsen with bending or lying down may raise suspicion for CVT due to increased ICP, though positional symptoms are neither sensitive nor specific for identifying CVT. Additional signs of intracranial hypertension include papilledema, nausea, vomiting, visual disturbances, and cranial nerve deficits.

Seizures are another common clinical feature of CVT, occurring in 20% to 50% of cases.[1,18,19] Clinicians should suspect CVT in patients with new or recurrent

unexplained seizures, particularly if preceded by headaches or associated risk factors. Focal neurologic deficits, seen in approximately 50% of cases, typically develop after headache onset and vary widely based on the infarct location. Neurologic deficits in CVT can include any constellation of transient vision changes (the most common of these deficits, seen in up to 27% of patients), cranial nerve deficits, hemiparesis, aphasia, and confusion, often lacking a clear, unifying pattern.[1,7,20]

Neurologic manifestations vary depending on the location of the thrombosis and the extent of infarction or hemorrhage; however, hemiparesis, aphasia, and other cortical signs are more common than cranial nerve deficits, which only occur in 6% to 11% of CVT patients.[1,7] Among cranial nerve deficits, the abducens nerve (CN VI) may be affected due to its long intracranial course and vulnerability to elevated ICP. Cranial nerves III, IV, VI, V1, and V2 may be affected in cases of cerebral sinus thrombosis.

A particularly challenging subset of patients includes those with altered mental status, encephalopathy, or coma. Confusion occurs in 31% to 43% of CVT cases and may be a prominent clinical feature, particularly in elderly patients and those with thrombosis of the deep venous system, including the straight sinus, internal cerebral veins, and vein of Galen.[21,22] Given the broad differential diagnosis for altered mental status, maintaining a high level of suspicion is critical for identifying CVT as the underlying cause.

The physical examination should include a thorough head-to-toe assessment to identify signs of systemic disease and hypercoagulable conditions, such as deep venous thrombosis, along with skin findings like petechiae, purpura, and livedo reticularis. A comprehensive ocular evaluation should be conducted to check for papilledema and evidence of oculomotor deficits related to cranial nerve dysfunction. Additionally, a complete neurologic examination is essential, documenting mental status, cranial nerve function, motor and sensory abilities, and cerebellar findings.

For an undifferentiated patient in the emergency department, any of the above signs and symptoms may prompt further evaluation for CVT. However, it is often the combination of risk factors and clinical features that guides the diagnosis. For instance, CVT should be suspected in patients with stroke-like symptoms, particularly if accompanied by atypical features such as a seizure or headache. It should also be considered in patients with stroke signs that cross typical arterial distributions. In cases without clear neurologic deficits, CVT should be considered in patients with headaches that feature atypical characteristics or risk factors, such as those who are pregnant or postpartum. Once a clinician has a high index of suspicion for CVT, further diagnostic workup is necessary.

Diagnosis

Imaging: The definitive diagnosis of CVT is confirmed through neuroimaging. In the emergency department, noncontrast computed tomography (NCCT) of the brain is typically the initial diagnostic choice due to its speed, availability, and ability to assess alternative diagnoses. However, NCCT has relatively low sensitivity, ranging from 41% to 73% for detecting cerebral venous thrombosis.[23,24] Positive findings on NCCT may suggest CVT but are not definitive.

Specific findings on NCCT can reveal direct signs of CVT, such as hyperdensity in a cortical vein or dural sinus. Ischemic infarctions may appear as edematous regions with mixed infarction, hemorrhage, and contrast enhancement crossing arterial boundaries. Indirect signs, such as a dense triangle or *filled delta sign* (**Fig. 2**) may indicate thrombosis in the posterior portion of the superior sagittal sinus.[7,23] Other indirect signs may include hemorrhage, which can be present in up to 40% of CVTs, as well as hydrocephalus or cerebral edema in cases of increased ICP.[1]

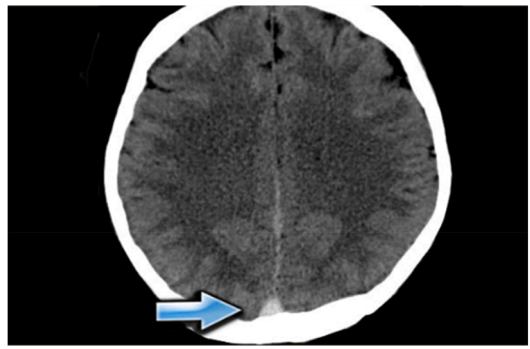

Fig. 2. Dense clot sign: also known as the "dense vein sign" refers to direct visualization of a hyperattenuating clot in the cerebral veins on a non-enhanced computed tomography scan. This sign is only visualized in approximately a third of cases. (Cerebral Venous Sinus Thrombosis. Retrieved from: https://radiologyassistant.nl/neuroradiology/sinus-thrombosis/cerebral-venous-thrombosis.)

Given the limitations of NCCT, the 2024 American Heart Association (AHA) CVT Scientific Statement recommends CT venography (CTV) or MR venography (MRV) as the optimal diagnostic tests, with MRV showing superior sensitivity, especially for cortical vein thrombosis.[1,24–26]

A CTV improves the diagnostic accuracy compared to NCCT alone with a sensitivity of approximately 95% and specificity of 91%.[27,28] CTV can reliably visualize a thrombus as a filling defect in the superficial and deep cerebral venous system. Thrombi can be identified as filling defects such as the *empty delta sign* (**Fig. 3**), which

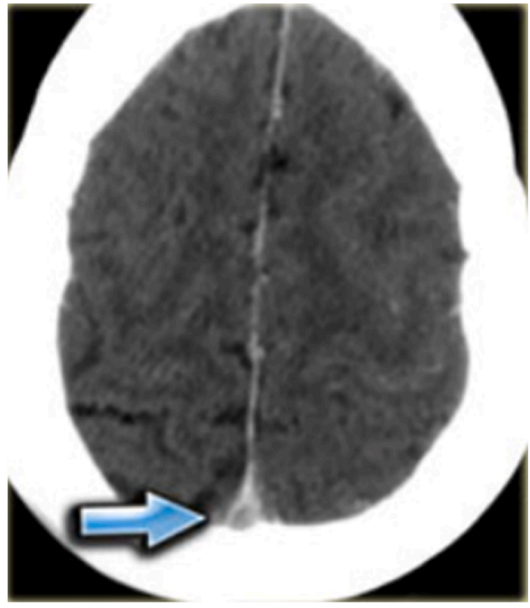

Fig. 3. Empty delta sign: a finding seen on contrast-enhanced computed tomography consisting of a triangular area of enhancement with a relatively low attenuating center, which is the thrombosed sinus. (Cerebral Venous Sinus Thrombosis. Retrieved from: https://radiologyassistant.nl/neuroradiology/sinus-thrombosis/cerebral-venous-thrombosis.)

refers to an opacification in the superior sagittal sinus. Contrast-enhanced MRV has a sensitivity and specificity comparable to that of CTV but demonstrates improved visualization of cortical vein thrombosis.[1]

Laboratory evaluation: Laboratory tests are generally unhelpful in the definitive diagnosis of CVT though testing may point to a potential underlying cause such as infectious and inflammatory causes or coagulopathies such as thrombophilia. Though D-dimers have demonstrated utility in the evaluation of deep venous thrombosis and pulmonary embolism, the role of D-dimer in the evaluation of cerebral venous thrombosis is less clear. Prior systematic reviews have demonstrated sensitivities of 93.9% and specificities of 89.7%, though a normal D-dimer should not be used to rule out CVT.[29]

Recent studies have explored the use of clinical prediction scores for CVT, though no tool is yet ready for clinical use. One study enrolled 359 patients from 2 emergency departments, aiming to combine D-dimer levels with clinical variables such as seizures, focal deficits, altered consciousness, or unexplained papilledema. The authors found that combining clinical markers with a specific D-dimer cutoff yielded an area under the curve of 0.94, but further external validation is needed.[30] Other researchers have retrospectively combined risk factors, clinical symptoms, and NCCT findings, showing high negative predictive values for ruling out CVT. However, these findings were derived retrospectively and require replication and validation before clinical consideration.[31]

Lumbar puncture (LP): While performing an LP to investigate alternative diagnoses such as meningitis or idiopathic intracranial hypertension, clinicians may encounter nonspecific CSF abnormalities in CVT patients. However, 1 prospective cohort study found that LP was negative in 44% of CVT patients. Current evidence does not support routine LP specifically for diagnosing CVT.[32]

Management

Initial stabilization: The emergency clinician's first priority when managing a CVT patient is stabilization and resuscitation, if needed. For critically ill patients with altered mental status or coma, airway assessment may reveal an inability to protect the airway or respiratory compromise, prompting consideration of endotracheal intubation. Depending on thrombus burden and affected areas, patients may present with seizures, requiring antiepileptic medications. Severe neurologic deterioration with suspected increased ICP should prompt interventions such as elevating the head of the bed $\geq 30^{\circ}$, hyperosmolar therapy, and targeted normocapnia.[33] If these measures are inadequate, emergent neurosurgical consultation may be necessary for more invasive treatments.

Medical management: Previously, bridging with low molecular weight heparin (LMWH) to vitamin K antagonists (VKAs) was considered the preferred treatment of CVT. However, recent head-to-head studies have shown that direct oral anticoagulants (DOACs) offer a similar safety and efficacy profile, making them a reasonable alternative to VKAs in most populations.[34–36] While the AHA still recommends LMWH bridging, emerging research questions whether a heparin bridge is necessary and, if so, the optimal duration.[1]

Surgical management: Neurosurgical consultation for endovascular treatment has not demonstrated a mortality benefit over medical management in severe CVT cases but may be considered for patients who deteriorate despite medical therapy or have contraindications to anticoagulation.[1] Further neurosurgical consultation for invasive interventions, such as decompressive craniectomy, may be offered for patients with impending herniation per AHA recommendations, though no randomized controlled trials currently support this practice.[1]

Special populations: Individual patient risk factors should guide the choice and duration of anticoagulation. For example, while CVT is a known complication of pregnancy and the puerperium, DOACs and VKAs are contraindicated during pregnancy and for breastfeeding patients.[1,37] Additionally, in patients with antiphospholipid syndrome, DOACs have been associated with a higher risk of recurrent thromboembolic events compared to VKAs.[38,39] Therefore, anticoagulation decisions should be made in close consultation with the admitting team, as well as neurology and hematology specialists.

Although extremely rare, documented cases of CVT and vaccine-induced thrombotic thrombocytopenia (VITT) have occurred in patients who received adenovirus-based COVID-19 vaccines. Due to molecular similarities to heparin-induced thrombocytopenia, current guidelines advise against using heparin in suspected VITT and CVT cases.

Disposition

Patients with CVT should be hospitalized for monitoring and initiation of anticoagulation, unless compelling reasons warrant alternative management.[7] Emergency providers should admit patients either to the ICU or to designated stroke units in consultation with neurology and hematology, and if those services are not available should be appropriately transferred for higher level of care.

Prognosis

CVT has a relatively low mortality rate of 5% to 10%, with up to 88% of patients achieving complete recovery or experiencing only mild functional or cognitive deficits.[40] The IN-REvASC study identified 7 key risk factors associated with poorer prognosis: active cancer, advanced age, Black race, encephalopathy or coma on presentation, decreased hemoglobin, higher NIHSS scores, and substance use.[40] While prior studies also highlighted sex, thrombus location, and birth control use as significant factors, these differences suggest the need for further research. Notably, pregnant women, once thought to have worse outcomes, are now understood to have similar or even improved prognoses compared to the general CVT population.[37]

Patients with CVT face an increased risk of recurrent venous thromboembolism, including recurrent CVT, particularly in the presence of severe thrombophilia. Beyond recurrence, long-term complications are increasingly recognized. Up to 50% of CVT patients report frequent headaches at follow-up, occurring at least once per week, while 1 in 5 experiences depression.[41] Additionally, cognitive impairments affect up to 34% of patients, though these deficits may not always be captured by standard functional assessments.[42] Further research is needed to better understand these chronic effects and develop early interventions aimed at improving long-term quality of life.

SUMMARY

CVT is a rare yet serious cerebrovascular condition distinct from arterial stroke. Despite its low prevalence (0.5%–3% of strokes), incidence has risen due to improved awareness and diagnostics. CVT presents variably, commonly with headache, seizures, and focal deficits, making early recognition crucial. Diagnosis relies on CT/MR venography, while anticoagulation remains the mainstay of treatment. Prognosis is generally favorable, though long-term complications like recurrent thrombosis and cognitive impairment are possible. Emerging research continues to refine diagnostic and management strategies, including anticoagulation choices and treatment of special populations such as pregnant patients.

CLINICS CARE POINTS

- Cerebral venous thrombosis (CVT) is a rare but serious cerebrovascular condition that remains difficult to diagnose due to its variable presentation and limited detection on standard brain imaging.
- Risk factors for CVT include hypercoagulable states, hormonal influences, infections, neurosurgical procedures, and emerging associations such as COVID-19 and vaccine-induced thrombocytopenia.
- Clinical presentation varies widely, with headaches being the most common symptom, followed by seizures, focal neurologic deficits, and altered mental status.
- Diagnosis relies on neuroimaging, with CT venography and MR venography being the most sensitive modalities.
- Management includes anticoagulation therapy as the mainstay of treatment, with direct oral anticoagulants emerging as a viable alternative to traditional heparin bridging; severe cases may require neurosurgical intervention.

DISCLOSURES

The authors have nothing to disclose.

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